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A CASE OF SPONTANEOUS CORONARY ARTERY DISSECTION (REVIEW AND CASE REPORT)

Abstract

The article presents a literature review on the problem of spontaneous coronary artery dissection (SCAD) — tearing of its wall, not associated with atherosclerosis, trauma or iatrogenic effects, leading to blood penetration between vessel layers of the artery. The consequence of this dissection is obstruction of the coronary artery due to the formation of intramural hematoma or intima damage and myocardial ischemia with development of acute coronary syndrome, myocardial infarction or sudden cardiac death. Information on the epidemiology, pathophysiology and etiology of the disease is presented in the paper. It highlights a role of arteriopathies, inflammatory diseases, pregnancy and female sex hormones, genetic causes as well as initiating and stress factors in SCAD development. The clinic picture and diagnosis of the disease are described. It was emphasized that in addition to clinical manifestations, the traditional electrocardiogram and coronary angiography remain the standard for diagnostics of the dissection. In the treatment of SCAD, percutaneous coronary intervention with stenting of the affected artery, coronary artery bypass graft and medications are used, with preference of conservative drug therapy. A special attention is paid to the features of diagnostic and therapeutic measures in pregnant and breast-feeding patients. The article also presents a clinical case of large-focal myocardial infarction complicated by cardiogenic shock in a young woman in the postpartum period without any risk factors for coronary heart disease, which was caused by SCAD. Diagnosis was accompanied by certain difficulties. An urgent percutaneous coronary intervention with stenting of the infarct-related coronary artery allowed to rapidly improve and stabilize the patient's condition.

Key words: *spontaneous coronary artery dissection, myocardial infarction, cardiogenic shock, diagnosis, coronary angiography, treatment, pregnancy, postpartum period*

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CABG — coronary artery bypass graft, SCAD — spontaneous coronary artery dissection, MI — myocardial infarction, IMH — intramural hematoma, LV — left ventricle, ACS — acute coronary syndrome, FMD — fibromuscular dysplasia, PCI — percutaneous coronary intervention, ECG — electrocardiogram

Introduction

Spontaneous dissection (rupture) of the coronary artery (SCAD) is a tear of its wall, not associated with atherosclerosis, trauma or iatrogenic effects, leading to the penetration of blood between the vessel membranes (tunica intima, tunica media and tunica externa). The consequence of such dissection is coronary artery obstruction due to the formation of intramural hematoma (IMH) or damage to intima and myocardial ischemia with development of acute coronary syndrome (ACS), myocardial infarction (MI) or sudden cardiac death [1].

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SCAD was described for the first time in 1931 [2], but in recent years, due to the rapid development of endovascular technologies, this problem has become particularly relevant [3].

Epidemiology

The true incidence of SCAD is unknown because of the difficulties with its diagnosis and the lack of experience among clinicians. However, it is believed that SCAD may cause ACS and MI in 1 to 4% of cases in the general population, in 35% of women aged under 50 years [4] and in 43% of pregnant women [5]. The left coronary artery is the most often affected (32–50%), and multiple SCAD occurs in 23% of cases [6].

Pathophysiology

SCAD is characterized by spontaneous formation of IMH within the coronary artery wall. This is confirmed by both intracoronary images and serial histopathological observations [4]. There are two theories for the development of SCAD. The first suggests that the primary pathological event is damage to the vascular wall (intima tear), which allows blood to leave the true channel of the vessel and create a false one. According to the second theory, spontaneous hemorrhage from own small artery vessels (vasa vasorum) within the vascular wall is considered to be the primary initiating event [7].

In the analysis of serial optical coherence tomography scans in patients with SCAD, it was noted that the site of intima damage can not always be found by this technique, which supports the second theory of pathogenesis [8]. In cases where it is possible to detect the site of damage to the intima, it remains unclear whether this is the initiating event or the consequence of increased pressure in the false channel, or the impact of the instrumental procedure on the coronary artery. The presence of an inflammatory cell infiltrate surrounding the dissection site can be used to recognize SCAD and to distinguish it from an iatrogenic one [9].

Etiology

The nature of SCAD seems to be multifactorial: hereditary or acquired arteriopathies, systemic inflammatory diseases, genetic defects, hormonal influences and their combinations with initiating stressful situations [10].

Arteriopathy

Among all arteriopathies, association of SCAD with multifocal fibromuscular dysplasia (FMD) is the most common (17–86%) [11]. This dysplasia, which affects any arterial bed, can manifest in the form of arterial stenosis, aneurysm, tortuosity or dissection. Multifocal FMD is its most frequent type and angiographically looks like zones of intermittent stenoses, dilations and aneurysms; the focal variant (<10% of cases) angiographically manifests itself as a single concentric or tubular narrowing. Currently, FMD is associated with conditions in which the fragility of arterial vessels is genetically mediated. Reports on typical histological and angiographic findings of FMD in the coronary arteries, including patients with previous events of SCAD, gave reason to believe that it can be a manifestation of a specific "coronary form" of FMD [12].

Pregnancy and female sex hormones

SCAD is the most common cause of MI in pregnant or postpartum female patients (approximately 1.81 cases per 100,000 pregnancies) [3]. The majority of its cases develop in the third trimester or early postpartum period, but there is evidence of SCAD in earlier gestation periods [13].

The causes of pregnancy-associated SCAD are not fully clear, although it is assumed that hormonal changes occurring during this period violate the architecture of the arterial wall, contribute to its rupture, the appearance of IMH and the onset of clinical symptoms [14]. The accumulation of these changes over several pregnancies may explain the increased risk of SCAD in women with multiple births. This is also facilitated by the presence of hypertension, lipid disorders, chronic depression, migraine, as well as age-related motherhood and infertility treatment in history [14].

Women with pregnancy-associated SCAD have a worse prognosis than other patients, and it is unclear why pregnancy-related disease is characterized by more aggressive widespread dissection and why it does not develop in most of them [15].

Inflammatory diseases

There are reports describing SCAD in patients with various systemic inflammatory processes, including systemic lupus erythematosus, inflammatory bowel disease, nodular periarteritis, sarcoidosis, and cryoglobulinemia secondary to hepatitis C [3].

Heredity and genetics

SCAD sometimes develops in individuals with hereditary arteriopathies and connective tissue dysplasia. Cases in patients suffering from Ehlers-Danlos vascular syndrome, Marfan and Loeys-Dietz syndromes, and also with polycystic kidney disease were described [16]. It is possible that there is a gene with a defect that increases the risk of SCAD, and studies are being conducted to identify it.

Initiating and stressful factors

Most often, SCAD is preceded by the release of catecholamines due to physical (24%) or emotional (40%) stress (as in stress-induced cardiomyopathy — Takotsubo syndrome). Emotional stress is more often observed in women, and physical stress — in men [17]. Hormonal changes associated with pregnancy, menopause, use of oral contraceptives, hormonal therapy, infertility treatment or high doses of corticosteroids also play a role [18].

Clinical picture

As a rule, in patients with SCAD there is almost always a picture of ACS and increased cardiac enzymes. Cardiogenic shock is seen in 2–5% of patients, the picture of ST-elevated MI – in 26–87%, without elevation — in 13–69%. Ventricular arrhythmias or sudden cardiac death develop in 3–11% of patients with SCAD [19].

Diagnosis

Diagnostic errors are often made in patients with SCAD, partly due to the young age of patients and the lack of risk factors for atherosclerosis. In case of suspected SCAD, it is **necessary to perform ECG** and coronary angiography as quickly as possible, especially when detecting ST segment elevation on the ECG [20]. Intravascular ultrasonography and optical coherence tomography provide a more detailed visualization of the vascular wall, which facilitates the diagnosis of SCAD, but they are not always available. Therefore, **traditional ECG and coronary angiography remain the standard for diagnosis of SCAD** [3].

Coronary angiography

According to J. Saw angiographic classification [12], type 1 SCAD is a classical manifestation of multiple radiolucent areas or defects in filling of arterial walls. Type 2 is characterized by the presence of diffuse stenosis which varies in severity and length (usually >20 mm): 2A variant is a diffuse narrowing of the artery, limited proximally and distally from IMH by normal segments, and 2B variant is a diffuse narrowing extending to the distal end of the artery. In case of type 3 there is focal or tubular stenosis, usually <20 mm in length, which mimics atherosclerosis. Naturally, intracoronary imaging allows to confirm IMH and SCAD.

It is shown [19] that the pattern of diffuse uniform stenosis (type 2) was the most frequent (67.5%) angiographic manifestation of SCAD, type 1 occurred in 29.1%, type 3 — in 3.4% of cases. It should be remembered that special care should be taken when performing coronary angiography in patients with SCAD, given the fragility of the coronary arteries and the risk of iatrogenic dissection.

Other imaging methods

Intravascular ultrasonography and optical coherence tomography are also used for diagnosis of SCAD, providing additional information, but have certain potential risks — aggravation (provocation) of coronary dissection by a probe or catheter, catheter-induced occlusion of the true artery lumen and hydraulic expansion by injection of false contrast [21].

Therefore, intracoronary imaging should only be performed when coronary angiographic diagnosis is unclear (type 3 or unknown injury) and when the vessel diameter is large enough for intracoronary imaging. Computer tomographic coronary angiography may also be a promising method for SCAD diagnosis [22].

Treatment

Although current guidelines for the management of patients with ACS of atherosclerotic origin recommend an early invasive strategy with revascularization of revealed disorders, there are no randomized studies with the results of revascularization in SCAD yet. Their necessity is crucial, since the mechanism of vascular obstruction, acute vascular response to balloon dilation and the natural outcome of conservatively treated catastrophes in SCAD differ significantly in comparison with ACS of atherosclerotic nature [3].

Conservative treatment

Despite the absence of comprehensive prospective studies, there is evidence that angiographic recovery of SCAD disorders is observed in the majority of patients (70–97%) who were re-examined weeks and months after their conservative management [21]. Persistent dissection was found in the minority of patients and it is unclear why it persisted. The length of recovery is also unclear, but there is evidence that it may take several days or weeks [17].

It should be remembered that early complications of recurrent MI associated with SCAD may develop in 5–10% of conservatively treated patients, mainly associated with increased dissection during the first week after an acute episode [14].

In high-risk patients with ongoing ischemia and dissection of the left coronary artery trunk or hemodynamic instability, percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) may be used.

Percutaneous coronary intervention

There is evidence that PCI in the treatment of SCAD is associated with an increased risk of complications and worse results due to the fragility of the arteries [17]. They are more susceptible to iatrogenic dissections, and coronary catheters often fall into the false lumen of the vessel and overlap the true one. Balloon dilation and stent placement may also increase the risk of vascular wall damage. In addition, the length of dissections is often extensive, which may require the use of longer coronary stents, which in turn increases the risk of subsequent intra-stent restenosis and thrombosis. In addition, SCAD most often affects the distal coronary segments, which may be too small or too remote for stent placement [23].

Therefore, in order to improve the outcomes of PCI in the case of SCAD, refrain from deep insertion of a catheter, non-coaxial placement of its tip, wetting the catheter, and a large injection of contrast agent. Instead of radial access, femoral access is preferred, which reduces the risk of catheter-induced iatrogenic dissection three times [19].

It is believed that for patients with SCAD in PCI the following are safer: (1) implantation of long drug-eluting stents that extend proximally and distally by 5–10 mm out of IMH areas to ensure their compression; (2) direct stenting without prior balloon dilation to avoid additional risks of IMH expansion; (3) isolated balloon angioplasty to restore coronary blood flow without stenting; (4) scoring balloon fenestration of IMH for decompression of blood portion from the false lumen to true one; (5) a multi-stent approach with initial stent sealing of the distal and proximal ends before stenting of median site in order to minimize IMH spreading; and (6) the use of bioresorbable stents to provide a temporary framework [23].

Naturally, after successful PCI, double antiplatelet therapy should be prescribed corresponding to stent implantation.

Coronary artery bypass graft

Published data on CABG in SCAD are limited to case descriptions, small series of observations, and retrospective analysis with a small sample size. CABG is described as a therapeutic strategy for SCAD in patients with dissection of the common left coronary artery trunk or the proximal segments of the arteries after a technical failure of attempt in case of PCI. At the same time, both arterial and venous shunts are used, although the long-term results of surgeries are not very comforting [20].

In summary, conservative therapy is generally the preferred strategy for the management of patients with SCAD who are clinically stable and have no objective signs of current ischemia, and this approach is generally associated with favorable outcomes. A conservative strategy is also appropriate in patients with distal vascular occlusion, which cannot be corrected in PCI [3].

Therapy after discharge from the hospital

Patients with SCAD who have undergone coronary revascularization should certainly receive *antiplatelet agents*, although there are no studies to assess its optimal duration and nature. Some experts recommend double antiplatelet therapy for at least a year, others — for a few months (1–3), followed by monotherapy with aspirin, which is taken for at least a year or indefinitely (if there are no contraindications) [24].
The use of *β-blockers* should be considered in patients with SCAD who have left ventricular (LV) dysfunction, arrhythmia, or hypertension, although some experts support the mandatory and long-term use of β-blockers [14].
Angiotensin-converting enzyme inhibitors or *angiotensin receptor blockers* should be prescribed if MI due to SCAD is complicated by LV dysfunction [25] or in concomitant hypertension. At the same time, women of reproductive age should be warned about the teratogenicity of antagonists of the renin-angiotensin system. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are contraindicated during gestation because their treatment in the 2nd and 3rd trimesters has been shown to be associated with toxic effects on the fetus (impaired renal function, oligohydramnios, decreased cranial vault, and sometimes fetal renal failure and even death) [26].
Statin therapy in SCAD is generally not used if there is no dyslipidemia, atherosclerosis or diabetes, and in the presence of anginal pain, nitrates, calcium channel blockers or ranolazine are used [3].

Spontaneous coronary artery dissection and pregnancy

Most pregnancy-associated SCAD cases occur in the first month after delivery, but they can develop in any gestational period [27]. The management of SCAD in such patients requires the interaction of cardiac and obstetric services [28]. However, despite the special situation in pregnancy, the principles of SCAD management are basically the same as without pregnancy, and in case of doubt in diagnosis and strategy, it is necessary to conduct early and careful angiography (with modern technologies, radiation for the fetus is relatively low [29]).
It should also be remembered that clopidogrel is not recommended for women who are breastfeeding, and small doses of aspirin are safe during pregnancy and breastfeeding. Despite the fact that β-blockers are associated with fetal growth restriction, they are often prescribed during pregnancy for the treatment of hypertension. Labetalol is preferred, especially in the early stages of pregnancy, and metoprolol and atenolol can result in reduced fetal weight and cause bradycardia in newborns during breastfeeding [30].
We have seen a case of SCAD in a young woman.

Case report

The patient, 35 years old, without any medical history, was delivered to the clinic by an ambulance team on 8.05.2018 with complaints of weakness, breathlessness and intense constricting pain in the upper chest, which abruptly occurred about 5 hours ago after a family dispute. On March 19, 2018, 50 days before the present deterioration, the patient gave birth to a healthy boy (cesarean section); the pregnancy period proceeded without complications.
Relatives of the patient first referred to the district doctor, who regarded these symptoms as manifestations of nervous and emotional stress and osteochondrosis and recommended the use of Valerian and ointment with diclofenac. But since the patient's condition did not improve, the relatives called an ambulance, and this doctor suspected her of acute coronary syndrome.
On admission: critical condition, adynamic, lethargic, responds to questions in monosyllables, with difficulty. She had normosthenic constitution The skin is pale, covered with cold sweat. Pulse is of small volume, arrhythmic, 106 min^{-1}, BP 80/40 mm Hg, the boundaries of the heart are normal, its tones are muffled, tachycardia, frequent premature beats. The thorax is painless during palpation, evenly participates in the act of breathing, pulmonary sound during percussion is normal throughout the surface, breathing is vesicular, 26 min^{-1}, there are no rales. The abdomen is soft, painless, from the navel to the pubis postoperative scar is seen. The lower edge of the liver is located at the line of costal arch; segments of the intestine are of normal properties.

She had childbirth by cesarean section 2 months ago in medical history (the child is not breastfeeding). There are no risk factors for coronary artery disease.

The patient's father had a dissecting aortic aneurysm, and the mother had two prior strokes.

On the ECG recorded by the doctor of the ambulance, there was elevated ST segment of 1 to 3 mm in leads V_2–V_5 (Fig. 1).

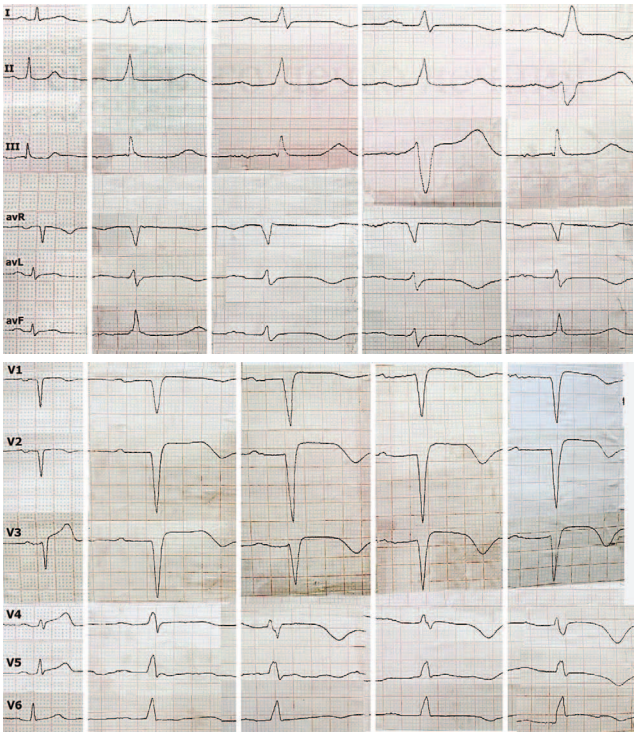
According to urgent echocardiography, there was akinesia of the apical anterior, apical septal, apical posterior; middle anterior septal LV segments, and hypokinesia of the middle anterior, middle posterior, septal LV segments.

Urgent coronary angiography (femoral access) revealed stenosis of up to 70% of the 1st portion of the anterior interventricular branch of the left coronary artery (left anterior descending artery, LAD) with transition to the ostium of diagonal branch and occlusion of the 2nd portion of LAD (acute thrombosis, TIMI flow grade 0) (Fig. 2).

During guidewire installation and balloon angioplasty, dissection of LAD (type 3 according to J. Saw angiographic classification) was found [12] (focal or tubular stenosis up to 20 mm in length, simulating atherosclerosis) (Fig. 3).

Stenting of LAD was performed (Fig. 4).

After PCI, the patient's condition immediately improved — pain in the chest disappeared, and blood pressure and pulse normalized (120/80 mm Hg). Subsequently, on the background of therapy with aspirin, clopidogrel, bisoprolol and lisinopril, it remained quite satisfactory. The patient well tolerated the expansion of the motor mode. Chest pain



On admission (paper speed 25 mm/sec)	1st day (50 mm/sec)	2nd day (50 mm/sec)	3rd day (50 mm/sec)	4th day (50 mm/sec)

Figure 1. *Dynamic electrocardiographic changes in a patient*

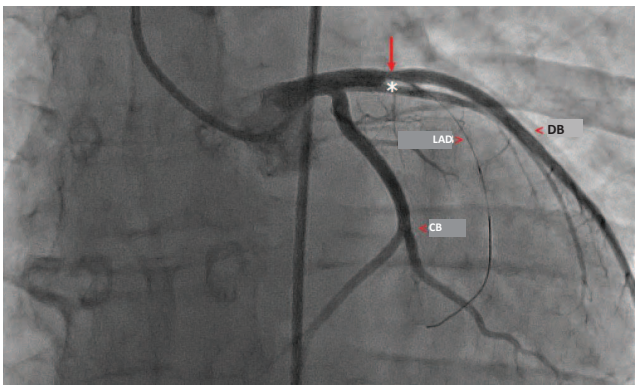


Figure 2. *Coronary angiography: stenosis of up to 70% of the 1st portion of the anterior interventricular branch of the left coronary artery (LAD) with transition to the ostium of diagonal branch (DB, marked by a red arrow) and occlusion (acute thrombosis type) of the 2nd portion of LAD (marked with a white asterisk), TIMI flow grade 0; a guidewire is seen in the artery (its line is marked by a small red arrow ">"); CB – circumflex branch*

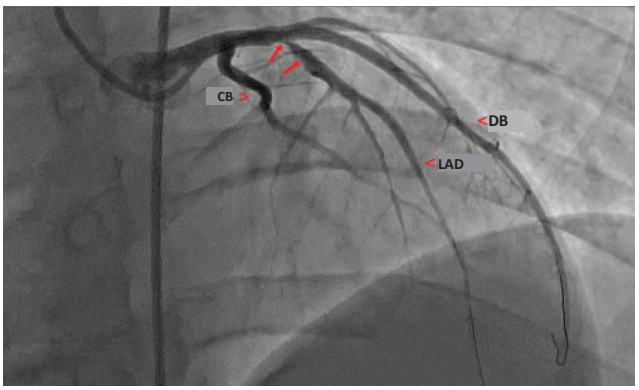


Figure 3. *The site of spontaneous coronary artery dissection after balloon angioplasty. Blood flow is partially restored along the anterior interventricular branch (LAD). Defects of the coronary artery wall are marked with red arrows. DB – diagonal branch; CB – circumflex branch*

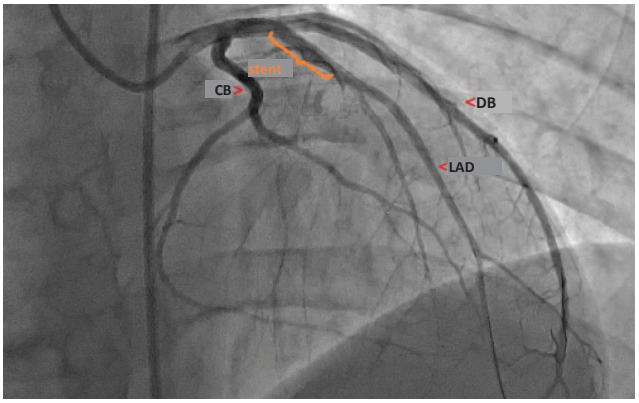


Figure 4. *Stenting of the anterior interventricular branch of the left coronary artery (LAD) with complete restore of blood flow. LAD stenting fragment is marked with an orange curly bracket. DB – diagonal branch; CB – circumflex branch*

and shortness of breath were not noted. On ECG series (Fig. 1) typical pattern, which is characteristic for acute large-focal anteroseptal LV MI, was observed. Two weeks later, the patient was discharged from the clinic with a recommendation to continue the therapy.

On the basis of clinical, anamnestic and ECG data, echocardiographic and angiographic studies, taking into account the course of the disease during treatment, the following diagnosis was made: Spontaneous dissection of the anterior interventricular branch of the left coronary artery. Acute large-focal MI in anterior and posterior septal segments and in the apex of the LV. Cardiogenic shock. Urgent PCI (balloon angioplasty and stenting of infarct-related artery) on 8.5.2018.

With control examination after 4 months: there were no complaints; in an objective status — without significant abnormalities. 6-minute walk test — 550 m.

Thus, in the young patient in the postpartum period, we observed acute large-focal LV MI caused by spontaneous dissection and thrombosis of LAD and the ostium of its diagonal branch, complicated by cardiogenic shock. Diagnosis was challenging. However, the clinical pattern, ECG recording and coronary angiography allowed to make the correct diagnosis, and urgent PCI quickly improved and stabilized patient's condition.

Conflict of interests

The authors declare no conflict of interests.

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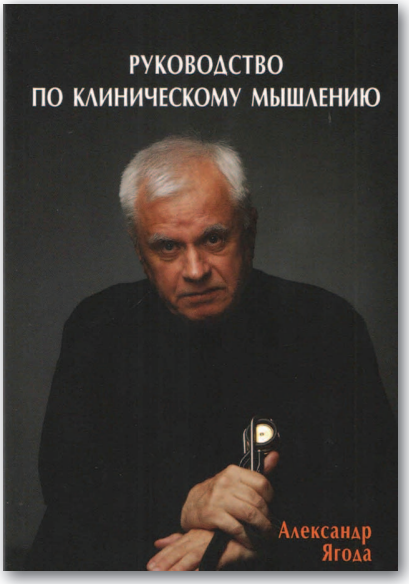

Уважаемые коллеги!

В издательстве «ЭКО Вектор» (С-Пб) в 2018 году вышла книга доктора медицинских наук, профессора, заведующего кафедрой госпитальной терапии Ставропольского государственного медицинского университета, заслуженного врача РФ, заслуженного деятеля науки РФ, члена редколлегии нашего журнала **Александра Валентиновича Ягоды** «РУКОВОДСТВО ПО КЛИНИЧЕСКОМУ МЫШЛЕНИЮ».

В книге с позиции автора в оригинальной форме клинических разборов, проводимых совместно со студентами в клиниках университета, на поликлиническом приёме сделана попытка донести до читателя методику мышления врача при опросе и осмотре больных, обсуждении данных лабораторного и инструментального исследований для выделения на этой основе главных, основополагающих позиций, на которых строится диагноз. Автор характеризует мышление врача как творческий процесс, базирующийся на теоретических знаниях, практическом опыте, логической деятельности ума, общей культуре и эрудиции, продолжая и развивая в своей работе гуманистические традиции Российской терапевтической школы. Книга адресована в первую очередь студентам старших курсов медицинских вузов, однако может быть полезна клиническим ординаторам и молодым врачам, а также всем, кто испытывает профессиональный интерес к клинике внутренних болезней.

Содержание: